

EVALUATION OF THE RES-Q-AIR
INHALATION REWARMING SYSTEM ON
NON-SHIVERING HYPOTHERMIC
SUBJECTS

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INHALATION REWARMING SYSTEM ON
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SUBJECTS**

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ABSTRACT

The objective of the present study was to test the efficacy of inhalation rewarming (IR) in simulated field conditions using a newly developed model of non-shivering hypothermia in humans. Eight subjects (2 of them female) were cooled in 8-10°C water for 25 min on two occasions. Ten minutes prior to withdrawal from the water, a total of 1.5 mg•kg⁻¹ of Meperidine, a shivering inhibitor, was injected intravenously into the subjects. The subjects were then removed from the water, settled in a sleeping bag and moved to a cold chamber set at -20°C for a 2.5 hour rewarming period. In the control trial, no external source of heat was available to the subject while in the IR trial, IR was provided (water saturated air at 43°C). In both trials, shivering was successfully suppressed since the metabolic rates during the rewarming phases were not different from baseline values. The duration and magnitude of the post-exposure decrease in esophageal temperature (afterdrop) were not different between the control (78 ± 11 min; 1.4 ± 0.1°C) and the IR trials (76 ± 10 min; 1.2 ± 0.2 °C). The rates of rewarming were not different between the two treatments (0.4 ± 0.1°C • h⁻¹ for control; 0.2 ± 0.1°C • h⁻¹ for IR). After the 2.5 hr rewarming phase, the esophageal temperature did not return to the pre-immersion level for either treatment (35.79 ± 0.17°C for control; 35.85 ± 0.13°C for IR). It was concluded that IR does not decrease the magnitude of the afterdrop and does not enhance rewarming rate in conscious non-shivering hypothermic subjects as compared to no external heat source.

EXECUTIVE SUMMARY

The Director of Medical Operations for the Canadian Forces (DMO 3-2) requested that DCIEM evaluate the Res-Q-Air inhalation rewarming system under operationally relevant ambient conditions. Inhalation rewarming is a technique proposed for active treatment of hypothermic victims in the field. Most studies that have evaluated inhalation rewarming treatment (IR) were performed at room temperature, and always with shivering mildly hypothermic subjects. The present study tested the Res-Q-Air IR system on non-shivering hypothermic human subjects during -20°C cold air exposure using a new model for severe hypothermia in humans.

Eight subjects (2 of them female) were cooled in $8-10^{\circ}\text{C}$ water for 25 min on two occasions. Ten minutes prior to withdrawal from the water, a shivering inhibitor was injected into the subjects. The subjects were then removed from the water, settled in a sleeping bag and moved to a cold chamber set at -20°C for a 2.5 hour rewarming period. In the control trial, no external source of heat was available to the subject while in the Res-Q-Air IR trial, IR was provided (water saturated air at 43°C). In both trials, shivering was successfully suppressed since the metabolic rates during the rewarming phases were not different from baseline values. No differences were observed between the control and the IR treatments for the duration and magnitude of the post-exposure decrease in esophageal temperature (afterdrop), the minimum temperature during the afterdrop, the rate of rewarming and the final esophageal temperature after 2.5 hours of rewarming. It was concluded that IR does not enhance the rewarming rate in conscious non-shivering hypothermic subjects as compared to no external heat source.

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INTRODUCTION

Lloyd in 1973 introduced for the first time an active non-invasive internal rewarming technique called inhalation rewarming (IR), which consists of providing warm (43-50°C) humidified air or oxygen to a hypothermic victim. Since then, despite numerous studies comparing IR to other rewarming techniques (Hayward and Steinman, 1975; Collis et al., 1977; Marcus, 1978; Morrison et al., 1979; Harnett et al., 1980; White et al., 1984; Romet and Hoskin, 1988; Sterba, 1991; Pozos et al., 1993; Mekjavik and Eiken, 1994), it has not been clearly established that airway rewarming alone can prevent or significantly reduce afterdrop, or can improve the rate of core rewarming (Harnett et al., 1983). Differences in the experimental procedures were held responsible for the reported discrepancies among studies (White et al., 1984; Romet and Hoskin, 1988; Sterba, 1991). Most studies that evaluated IR were performed at room temperature and always using shivering hypothermic subjects. Those studies do not mimic the conditions prevalent in a severe case of accidental hypothermia in the field, where the shivering is suppressed in the victim and active rewarming techniques are the most useful. Harnett et al. (1983) stated in their review of literature concerning resuscitation from hypothermia that "most resuscitation measures should be aimed at reviving hypothermia victims with rectal temperatures below 32°C" when shivering can be impaired. The large heat production from shivering in a mild hypothermic victim decreases the ability to differentiate between other external methods of rewarming. When subjects are deeply hypothermic, shivering progressively disappears and rewarming techniques can become effective to rewarm the patients. To what extent IR is effective in rewarming hypothermic non-shivering patients is not known. Our intent was to test the efficiency of IR in simulated field conditions (-20°C) using a newly developed model of non-shivering hypothermia in humans (Giesbrecht et al., 1995).

The University of Victoria, British Columbia, has recently designed and developed a new version of an inhalation rewarming device. The Model HT 1000 Inhalation Delivery System (Res-Q-Air, C.F. Electronics Inc., New York, U.S.A.) is said to be a substantial improvement over previous models, being safer, easier to use, and having very good temperature control. Indeed, several unsafe characteristics of the earlier model, the Heat Treat (Thermogenesis, Victoria, British Columbia) were reported by Sterba (1991), such as improper ignition of the propane canister, excessive temperature of the unit, difficult temperature control of the inhalation temperature, and air flow restriction. The Director of Medical Operations for the Canadian Forces (DMO 3-2) requested that DCIEM evaluate the

new inhalation rewarming device for safety and performance, and to re-evaluate the system under operationally relevant ambient conditions.

The objectives of the present study were, therefore, to evaluate the safety and efficiency of the Res-Q-Air Inhalation Delivery System as a first-aid treatment device for stabilizing / rewarming hypothermia victims under cold ambient conditions, and to make appropriate recommendations to the CF. The present report focusses on the performances of the Res-Q-Air IR treatment during tests performed on non-shivering hypothermic subjects. Due to the limitations of the non-shivering hypothermic model, the testing was performed only on mildly hypothermic non-shivering subjects.

MATERIAL AND METHODS

Subjects. Eight healthy subjects (6 males and 2 females) volunteered to participate in the study. Their anthropometric characteristics are presented in Table 1. The percentage of body fat was estimated from the summation of four skinfold thicknesses (triceps, biceps, suprailiac and subscapular) measured by a Harpenden skinfold caliper (British Indicator, England) and calculated using the relationship developed by Durnin and Womersley (1974). The health status of all subjects was assessed by a medical authority before participation. The subjects were fully informed of the procedures and possible risks of the study and their right to withdraw from the experiment at any time without prejudice. Written informed consent was obtained from all subjects before experimentation. The protocol was approved by Institutional Ethics Committees.

The subjects were asked to abstain from smoking and using any medication, drug, or other stimulant (including caffeine and alcohol) for at least 12 h before the experiments. All experiments were performed at the same time of the day for each subject.

Temperature and heat flow measurements. Ambient temperature was continuously measured outside (cold chamber) and inside the sleeping bag using type T thermocouples. Three temperature probes located in air 2 to 4 cm from the skin of the subjects were used to measure the micro-environment inside the sleeping bag above the thigh and the chest, and under the lower back of the subjects.

Skin temperature (T_{skin}) using type T thermocouples (integrated into the heat flux transducers) and skin heat loss (\dot{H}_{skin}) using heat flux transducers (HFTs, Concept

Table 1. Anthropometric characteristics of the subjects

Subject #	Age, yr	Height, cm	Weight, kg	A _D m ²	Skinfold thickness mm	Body fat %
1*	23.1	172	67.3	1.79	113.8	38.3
2	37.2	183	82.0	2.04	62.6	23.9
3	30.6	176	82.5	1.99	38.6	18.8
4	31.2	181	72.0	1.92	27.8	15.3
5	25.2	184	75.5	1.97	40.3	16.5
6*	34.5	161	63.7	1.67	67.5	32.0
7	26.2	176	71.0	1.86	27.4	11.7
8	35.8	185	80.0	2.03	49.6	18.9
mean \pm SE	30.5 \pm 1.8	177 \pm 3	74.3 \pm 2.5	1.91 \pm 0.05	53.5 \pm 10.1	21.9 \pm 3.2

A_D, DuBois surface area (Dubois and Dubois, 1916); skinfold thickness represent the summation at four sites: triceps, biceps, suprailiac and subscapular; *, female subject.

Engineering, Old Saybrook, CT) were measured on 12 sites according to the Hardy and Dubois modified 12 points weighting system (Olesen, 1984). The HFTs were recalibrated according to the method of Ducharme et al. (1990) and the heat flow values were corrected to account for the thermal insulation of the HFTs (Ducharme et al., 1990).

Core temperature of the subject was measured from 3 different sites: esophagus, rectum, and left ear canal. Esophageal temperature (*T_{es}*) was measured with a type T thermocouple (Mon-a-therm General Purpose, Mallinckrodt Medical, St. Louis, MO) positioned at the level of the heart using the method of Mekjavik and Rempel (1990). Rectal temperature (*T_{re}*) was measured with a type T thermocouple (Mon-a-therm General Purpose, Mallinckrodt Medical, St. Louis, MO) positioned 15 cm into the rectum. Left ear canal temperature (*T_{ear}*) was measured with a fine type T thermocouple (Mon-a-therm Tympanic temperature sensor Mallinckrodt Medical, St. Louis, MO) positioned very close to the tympanic membrane (after touching the tympanic membrane, the probe was withdrawn just enough for the pain to disappear). The outside portion of the ear canal was filled with cotton and tape was used over the auricle to fix the probe and insulate the ear canal from the environment.

Serial data from the HFTs and the thermocouples were acquired continuously during the cooling and rewarming phases on an electrically isolated Macintosh IIfx computer and averaged every 30-s period. The process was controlled by a "virtual instrument" written using LabVIEW II graphical signal processing software (National Instruments, Austin, TX).

Metabolic heat production, heart rate, and blood pressure measurements. Oxygen consumption ($\dot{V}O_2$) was measured continuously during the resting, cooling and rewarming phases with an open-circuit method (O_2 analyser: Beckman model OM-11, Beckman, Anaheim, CA) from measurements of expired minute volume and inspired and mixed expired gas concentrations sampled from a mixing box. Subjects wore a snugly fitting face mask (Hans Rudolph Inc, Kansas City, MO, USA.) with a one-way valve, which was connected to a flow transducer (Hewlett Packard model 47304A) and the mixing box. Oxygen consumption was converted to heat production (\dot{M} in $W \cdot m^{-2}$) using the following equation regressed from McArdle et al. (McArdle et al., 1981):

$$\dot{M} = (266 + 86 \cdot RQ) \dot{V}O_2 / A_D$$

where RQ is the respiratory quotient assumed to be mixed at 0.82, $\dot{V}O_2$ is the rate of O_2 uptake ($L \cdot min^{-1}$), and A_D is the body surface area (m^2).

Heart rate and ECG were continuously monitored on a monitor defibrillator (Hewlett Packard model 43100A). Blood pressure was continuously monitored with an automated blood pressure monitoring system (Dinamap model 845XT, Critikon Inc, Tampa, FL), and mean arterial pressure (MAP) was measured by the monitoring system at the peak oscillation in the cuff pressure.

Procedures. Subjects were cooled on two occasions separated by a week. Before the immersion in water, the subjects were instrumented with i) a disposable and sterile rectal probe, ii) a disposable and sterile esophageal probe, iii) ECG leads for continuous cardiac monitoring, iv) an ear canal temperature probe in the left ear, v) 12 heat flow transducers (incorporating skin temperature sensors) taped on the skin of the forehead, scapula, chest, abdomen, lower back, lower arm, hand, anterior and posterior thigh, calf, shin and foot according to the modified Hardy & Dubois weighting system (1), vi) a venous catheter on the arm (with an external line extending to the neck of the subject) for injection of drugs, and vii) a thin plastic body suit covering all parts of the body up to the neck (this suit allowed the subject to stay dry during the water immersion which minimized

evaporative heat loss upon exit from the water). The resting metabolism was then measured for a period of 10 min while sitting at thermal neutrality.

The subject was then lifted by a crane while sitting on a nylon harness and lowered into the cold water bath, staying in water until i) rectal temperature reached 36.5°C (the efficiency of the shivering inhibitor Meperidine (Demerol®) is temperature sensitive; this rectal temperature was chosen to optimize the action of the drug), ii) the subject asked to be removed from the water or iii) the attending physician or investigators ended the exposure. The temperature of the water upon entry was approximately 20-22°C. Following entry, ice was added to the stirred water to lower the temperature of the water over the next 10 min to approximately 8-10°C. Shortly before exiting the water, Meperidine, a central depressant which inhibits shivering, was infused through an arm vein at 20 mg every 2 min to a maximum of 1.0 mg/kg body weight. Thereafter, Meperidine was infused at 10 mg as required to suppress shivering to the maximum cumulative dose of 1.5 mg/kg body weight. Note: this is a maximum dose and infusion was ceased if respiratory depression or other untoward side effects occurred. To ensure the well being of the subjects, blood pressure and oxygen saturation at the finger tip (Ohmeda Pulse Biox model 3700 Pulse Oximeter, Ohmeda Inc., Louisville, CO) were continuously monitored from the injection time. Following the immersion, the plastic body suit was rapidly removed and the subject was fitted with long thinsulate insulated socks (up to the knee) and mitts (up to the elbows). The subject was then put onto a suspended webbing bed inside an Arctic sleeping bag fixed on a stretcher. The purpose of the insulated socks and mitts was to minimize the cold venous blood return from the extremities to the core of the subjects during the rewarming period and therefore to minimize the afterdrop (a further decrease of core temperature following removal from the cold stress). An insulated hood covered the head of the subjects. The subject was then moved into a cold chamber set at -20°C air temperature for the rewarming phase. Efforts were taken to minimize the magnitude of the afterdrop not related to the rewarming treatments; these included using a crane to remove the subject from the water, reducing muscular effort or rough handling during the transfer, and the use of the plastic suit to eliminate skin wetting and evaporative heat loss.

One of the following two rewarming procedures was tested randomly: control and IR with Res-Q-Air. The rewarming phase lasted 2.5 hours or i) until the core temperature of the subjects has rewarmed to 36.8°C (which is considered normothermic; Mackowiak et al., 1992) or ii) in case of ineffective rewarming, until rectal temperature reached 33.5°C, or iii) until the subjects asked to terminate the rewarming procedure (the subjects still had to be rewarmed to normal body temperature in a hot bath) or iv) the attending physician or investigators ended the rewarming phase. During the control rewarming, no non-

endogenous heat source was provided to the subjects and shivering was suppressed by the action of the Meperidine. The only endogenous heat source available to the subjects for rewarming was their resting metabolism. During the IR rewarming the outlet hose of the Res-Q-Air unit (Res-Q-Air, model HT 1000, C.F. Electronics Inc. Commack, NY) was fixed to the inlet port of the breathing mask used for $\dot{V}O_2$ measurement. The subjects were breathing water-saturated air at $42.9 \pm 0.4^\circ\text{C}$ from the Res-Q-Air system. The temperature of the breathing air was confirmed by a type T thermocouple fixed inside the mask at the inlet port.

If the core temperature of the subjects at the end of the rewarming period was not restored to a normothermic value of 36.8°C , the subjects were rewarmed in a hot bath filled with water at $38\text{--}40^\circ\text{C}$ until core temperature reached 36.8°C .

Data analyses: Evaluation of the Res-Q-Air inhalation system. The following variables were calculated for the control and the IR trials: the afterdrop (defined as the difference between *Tes* at the end of the immersion and its nadir following the immersion); length of the afterdrop period (defined as the time from exiting cold water until *Tes* returned to the original post-immersion *Tes*); rewarming rate (calculated from the linear increase following the *Tes* nadir until the end of the rewarming period); mean skin heat loss and mean skin temperature, and metabolic heat production. Data for the two trials were compared using paired t-test (Statview software, Abacus Concepts Inc., Berkeley, CA, 1992). Results are reported as 10 minute means \pm SE (except for the nadir temperatures during the rewarming phases which are 30 second averages) and differences were considered significant when $p < 0.05$.

RESULTS

The dose of Meperidine used in the present study did not induce symptoms of overdose such as a decrease in systolic blood pressure or a suppression of the respiratory function.

Physiological measurements during the evaluation of the Res-Q-Air inhalation system. During the rewarming phase of the trials, the cold room was maintained on average at $-18.3 \pm 0.3^\circ\text{C}$ and the microclimate inside the sleeping bag remained at $22.5 \pm 0.4^\circ\text{C}$ due to body heat. No difference was observed between treatments.

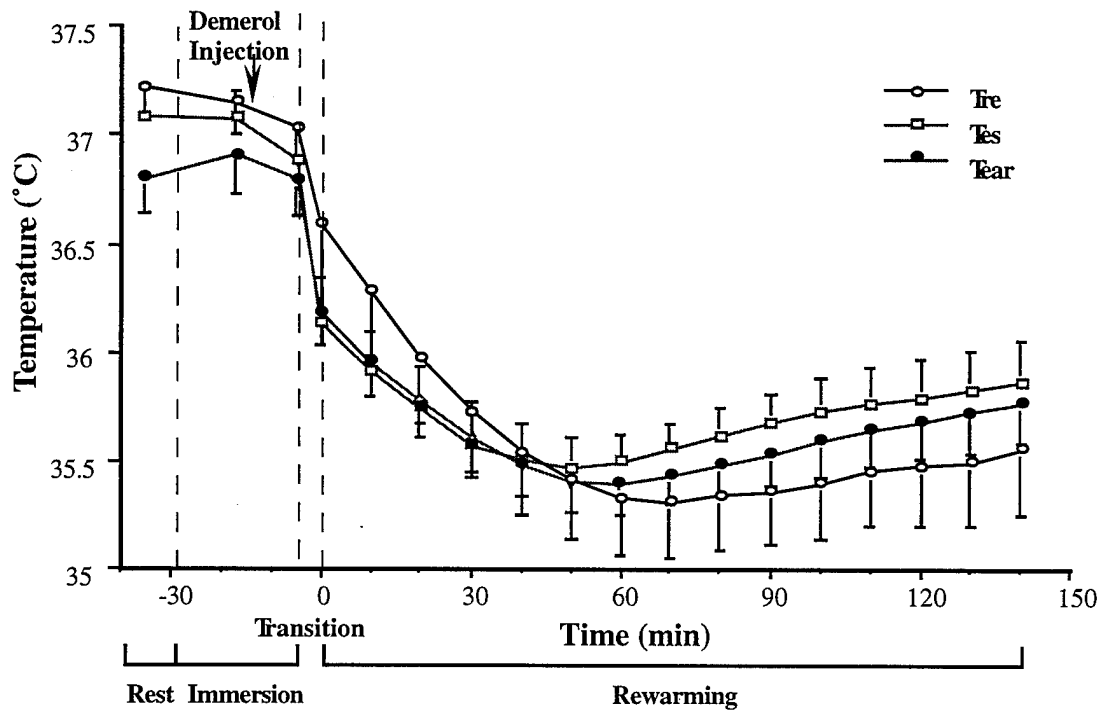


Figure 1. Rectal (T_{re}), esophageal (T_{es}) and ear canal temperatures (T_{ear}) observed during the different phases of the control trial (see Methods for detailed procedures). $n=8$, mean \pm SE.

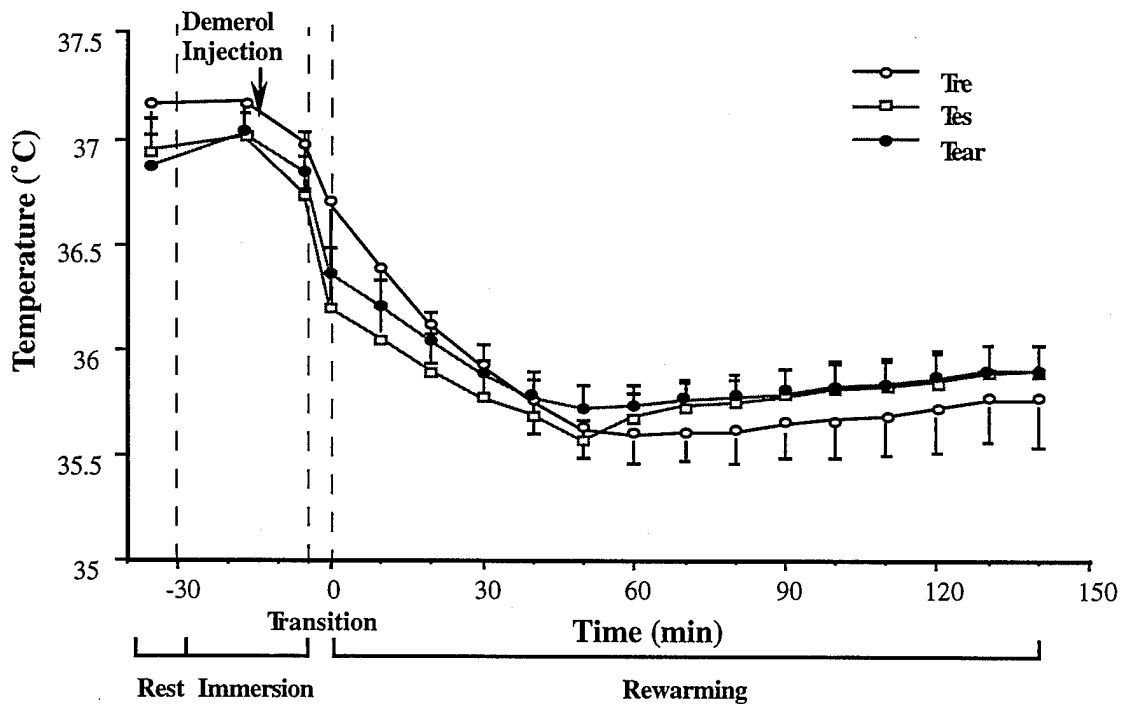


Figure 2. Rectal (T_{re}), esophageal (T_{es}) and ear canal temperatures (T_{ear}) observed during the different phases of the Inhalation Rewarming (IR) trial (see Methods for detailed procedures). $n=8$, mean \pm SE.

Core temperature. For the evaluation of the Res-Q-Air system, esophageal temperature (*Tes*) was used to represent core temperature since *Tes* is a good estimation of mixed blood temperature (Lloyd, 1986) and is the fastest responding site to warmth and cold (Houdas and Ring, 1982). The three core temperature indices (*Tes*, *Tear* and *Tre*) however, followed similar patterns, *Tes* being normally between *Tre* (highest) and *Tear* (lowest) in a normothermic subject and lower than *Tre* and *Tear* during the afterdrop of the rewarming phase (see Figs. 1 to 5).

On average, *Tes* measured during the 10 min resting period preceding the immersion (baseline temperature) was $37.02 \pm 0.15^{\circ}\text{C}$ and no difference was observed between the control and IR treatments. In addition, no difference was observed in the immersion time between the two treatments (control: 25.9 ± 4.1 min; IR: 25.7 ± 4.4 min). The afterdrop, defined as the difference between *Tes* on exit from the cold water and its nadir, was not different between the control ($1.40 \pm 0.08^{\circ}\text{C}$) and IR ($1.22 \pm 0.16^{\circ}\text{C}$) treatments, and neither was the time to reach the *Tes* nadir (control: 77.9 ± 11.5 min; IR: 75.8 ± 9.9 min). *Tes* nadir, defined as the lowest recorded temperature during the rewarming phase based on 30 s averages, was not different between the control treatment ($35.37 \pm 0.15^{\circ}\text{C}$) compared to the IR treatment ($35.25 \pm 0.16^{\circ}\text{C}$). The rate of rewarming, calculated by linear regression for *Tes* data, was not different between the control ($0.41 \pm 0.13^{\circ}\text{C} \cdot \text{h}^{-1}$) and IR treatment ($0.23 \pm 0.05^{\circ}\text{C} \cdot \text{h}^{-1}$). By the end of the 2.5 hrs rewarming period, *Tes* did not return to pre-immersion level for either treatment. *Tes* after the rewarming period was on average $35.82 \pm 0.15^{\circ}\text{C}$ and no difference was observed between treatments (control: $35.79 \pm 0.17^{\circ}\text{C}$; IR: $35.85 \pm 0.13^{\circ}\text{C}$; see Fig. 3).

It was found that the warm air delivered by the Res-Q-Air unit did not directly affect the *Tes* readings. During the rewarming phase using Res-Q-Air system, *Tes* decreased to a lower nadir value ($35.25 \pm 0.16^{\circ}\text{C}$) than either *Tre* ($35.62 \pm 0.11^{\circ}\text{C}$) or *Tear* ($35.65 \pm 0.11^{\circ}\text{C}$). Thereafter, for the rest of the rewarming phase, *Tes* resumed its "middle" position between *Tre* and *Tear* (see Fig.2; note that the data are expressed as 10 min averages).

Heat flow and skin temperatures. In every phase of the trials, heat flow was not different between the control treatment and the IR treatment. Heat flow increased from an average baseline level of $82.1 \pm 6.7 \text{ W} \cdot \text{m}^{-2}$ to a peak value of $413.8 \pm 37.2 \text{ W} \cdot \text{m}^{-2}$ before injection of the Meperidine, and thereafter decreased to an average of $308.4 \pm 42.0 \text{ W} \cdot \text{m}^{-2}$ just before the exit from the water (see Fig. 6). During the rewarming phase, the heat flow averaged $45.9 \pm 3.9 \text{ W} \cdot \text{m}^{-2}$.

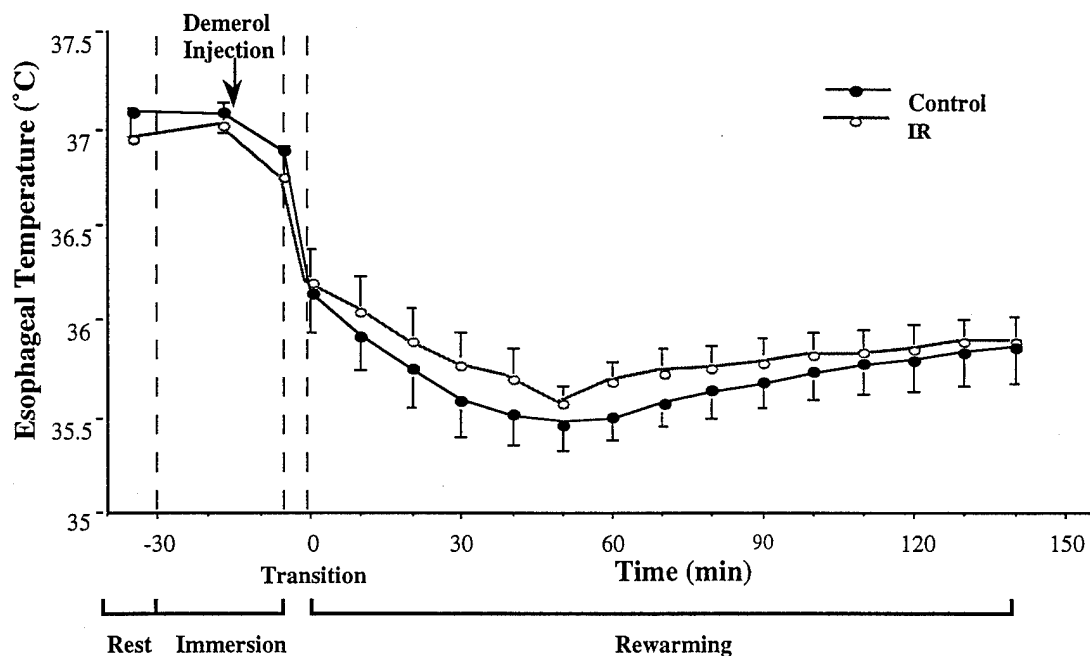


Figure 3. Esophageal temperature (T_{es}) observed during the different phases of the Control and Inhalation Rewarming (IR) trials (see Methods for detailed procedures). $n=8$, mean \pm SE.

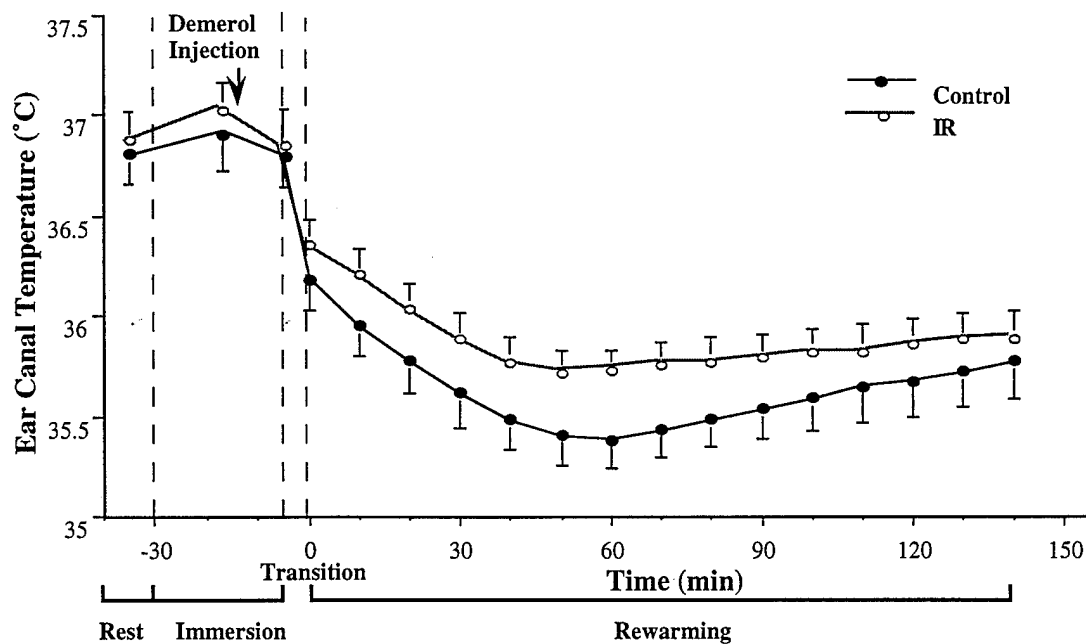


Figure 4. Ear canal temperature (T_{ear}) observed during the different phases of the Control and Inhalation Rewarming (IR) trials (see Methods for detailed procedures). $n=8$, mean \pm SE.

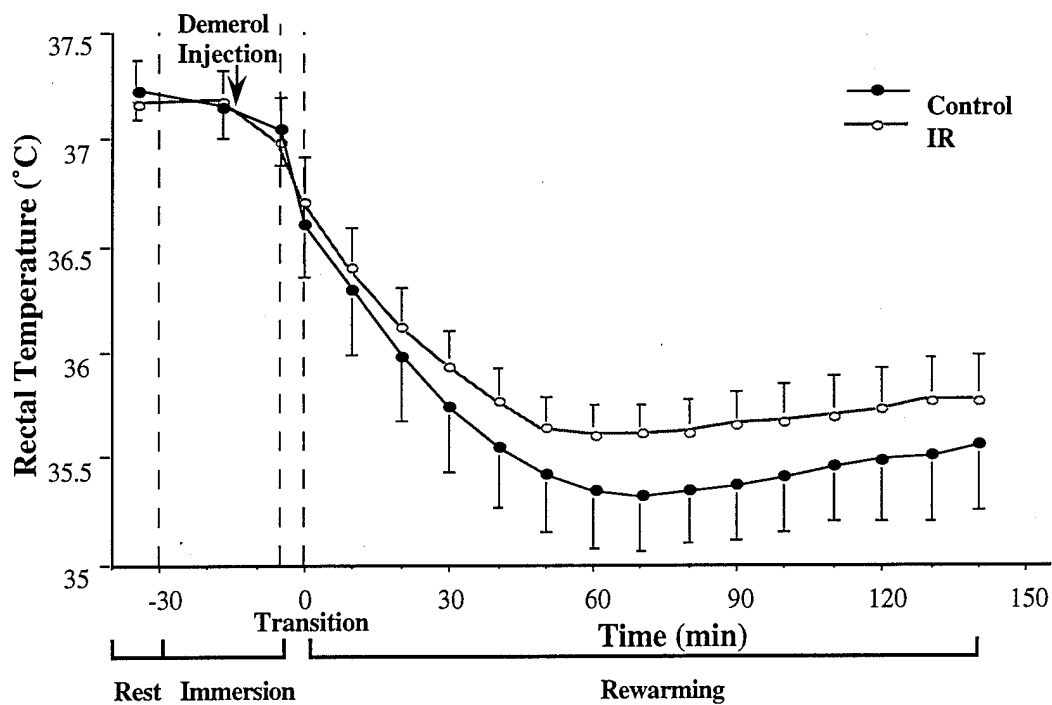


Figure 5. Rectal temperature (T_{re}) observed during the different phases of the Control and Inhalation Rewarming (IR) trials (see Methods for detailed procedures). $n=8$, mean \pm SE.

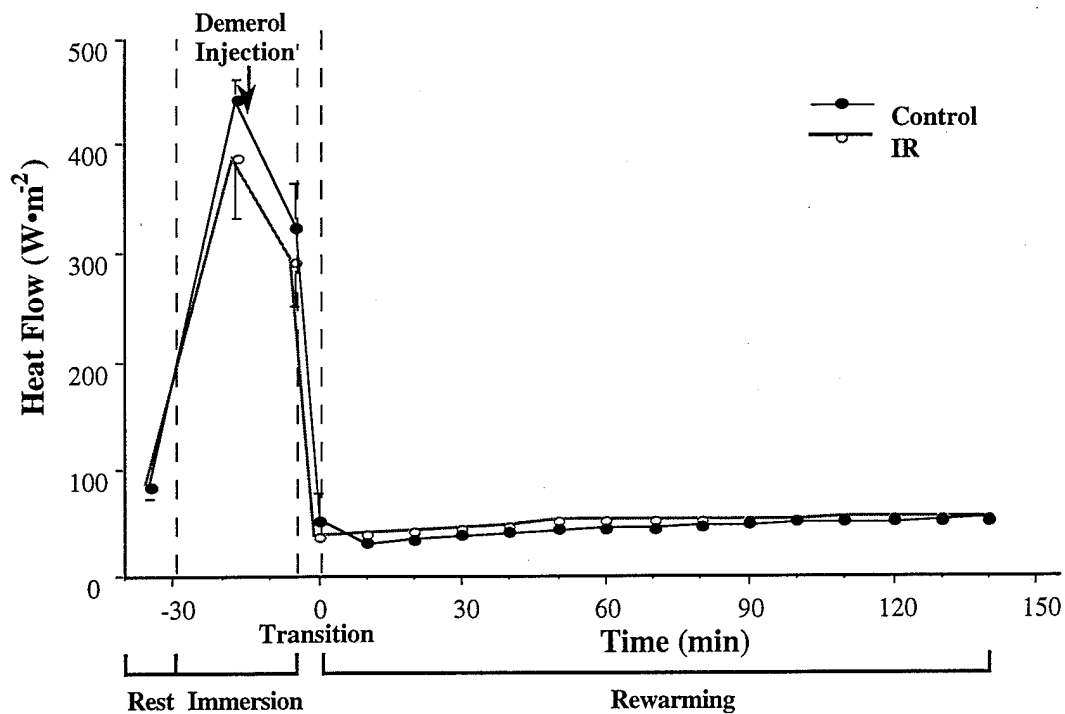


Figure 6. Mean skin heat loss (H_{skin}) observed during the different phases of the Control and Inhalation Rewarming (IR) trials (see Methods for detailed procedures). $n=8$, mean \pm SE.

Mean skin temperature (T_{sk}) was also not different between the control and the IR treatments. T_{sk} decreased from an average baseline level of $31.66 \pm 0.22^{\circ}\text{C}$ to $20.70 \pm 1.23^{\circ}\text{C}$ before injection of the Meperidine and to $19.69 \pm 0.94^{\circ}\text{C}$ by the end of the water immersion. During the rewarming phase, T_{sk} increased significantly to an average of $28.96 \pm 0.51^{\circ}\text{C}$ by the end of the rewarming period (see Fig. 7).

Cardiovascular and metabolic responses. No difference was observed in heart rate (HR) between the control and IR trials. HR increased significantly from an average baseline value of $73.3 \pm 4.8 \text{ beat} \cdot \text{min}^{-1}$ to $79.5 \pm 5.4 \text{ beat} \cdot \text{min}^{-1}$ just before the injection of Meperidine, and thereafter decreased to an average of $67.1 \pm 4.1 \text{ beat} \cdot \text{min}^{-1}$ just prior to water exit. During the rewarming phase, HR continued to decrease to a value of $52.8 \pm 2.7 \text{ beat} \cdot \text{min}^{-1}$ (see Fig. 8).

No difference was observed in mean arterial pressure (MAP) between the control and IR. MAP increased significantly from an average baseline value of $92.5 \pm 1.8 \text{ mmHg}$ to $110.0 \pm 3.1 \text{ mm Hg}$ just before the injection of Meperidine, and then decreased to an average of $106.3 \pm 2.7 \text{ mm Hg}$ prior to water exit. During the rewarming period, MAP continued to decreased to an average value of $94.6 \pm 3.0 \text{ mm Hg}$ (see Fig.9).

No difference was observed in oxygen consumption ($\dot{V}O_2$) for the baseline and cooling period between the control and IR treatments. $\dot{V}O_2$ on average increased from baseline value of $325.8 \pm 21.8 \text{ ml} \cdot \text{min}^{-1}$ to $566.5 \pm 47.0 \text{ ml} \cdot \text{min}^{-1}$ just before the injection of Meperidine, and decreased to an average of $365.0 \pm 28.6 \text{ ml} \cdot \text{min}^{-1}$ prior to water exit. During the rewarming period, the $\dot{V}O_2$ for the IR treatment was significantly higher ($359.9 \pm 23.8 \text{ ml} \cdot \text{min}^{-1}$) than control $\dot{V}O_2$ ($297.4 \pm 23.1 \text{ ml} \cdot \text{min}^{-1}$). The difference in $\dot{V}O_2$ was the largest at 30 min into the rewarming phase (see Fig. 10). On average for both treatments, $\dot{V}O_2$ during the rewarming period was not different from baseline $\dot{V}O_2$. This implies that Meperidine was effective in suppressing shivering during the rewarming phase.

The minute ventilation ($\dot{V}e$) showed a trend similar to $\dot{V}O_2$. $\dot{V}e$ on average increased from baseline value of $14.3 \pm 1.8 \text{ L} \cdot \text{min}^{-1}$ to $23.5 \pm 3.0 \text{ L} \cdot \text{min}^{-1}$ just before the injection of Meperidine, and decreased to an average of $14.4 \pm 2.3 \text{ L} \cdot \text{min}^{-1}$ prior to water exit. During the rewarming period, there was a tendency for the $\dot{V}e$ during the IR treatment to be higher ($13.2 \pm 3.3 \text{ L} \cdot \text{min}^{-1}$) than control $\dot{V}e$ ($9.5 \pm 0.6 \text{ L} \cdot \text{min}^{-1}$), but the difference was not significant (see Fig.11).

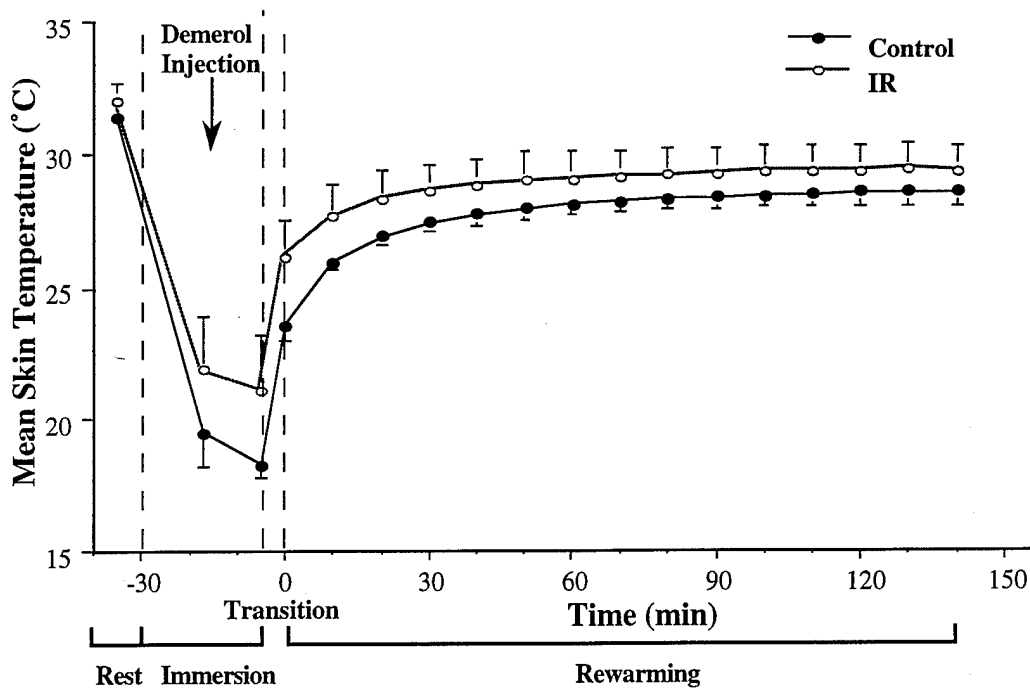


Figure 7. Mean skin temperatures (T_{sk}) observed during the different phases of the Control and Inhalation Rewarming (IR) trials (see Methods for detailed procedures). $n=8$, mean \pm SE.

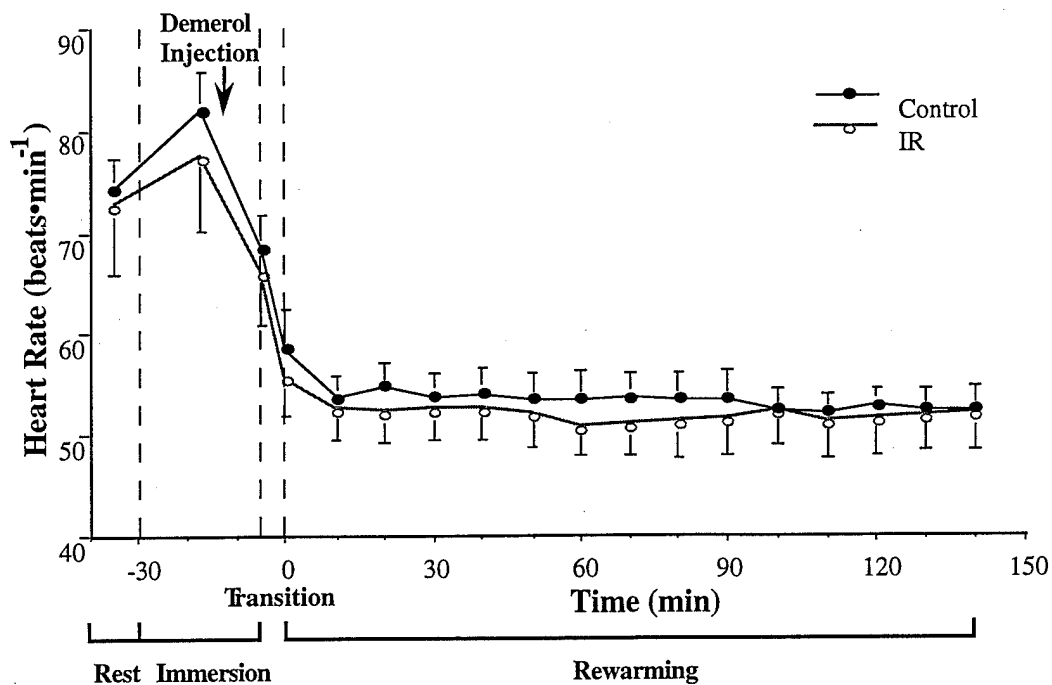


Figure 8. Heart rate (HR) observed during the different phases of the Control and Inhalation Rewarming (IR) trials (see Methods for detailed procedures). $n=8$, mean \pm SE.

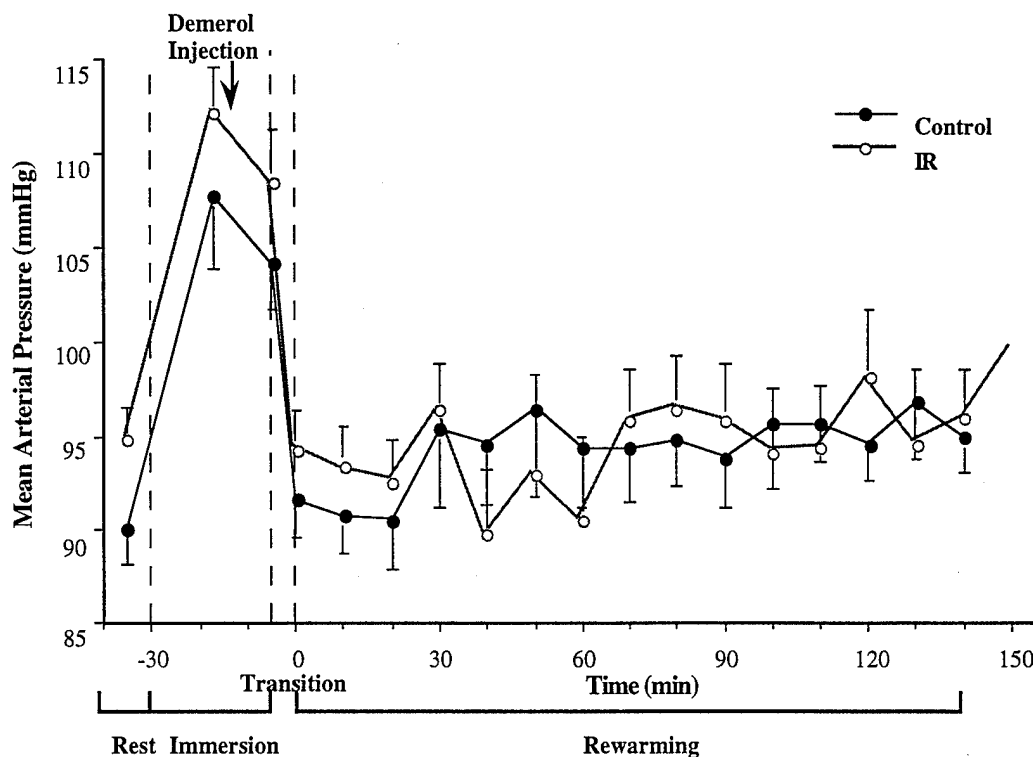


Figure 9. Mean arterial pressure (MAP) observed during the different phases of the Control and Inhalation Rewarming (IR) trials (see Methods for detailed procedures). $n=8$, mean \pm SE.

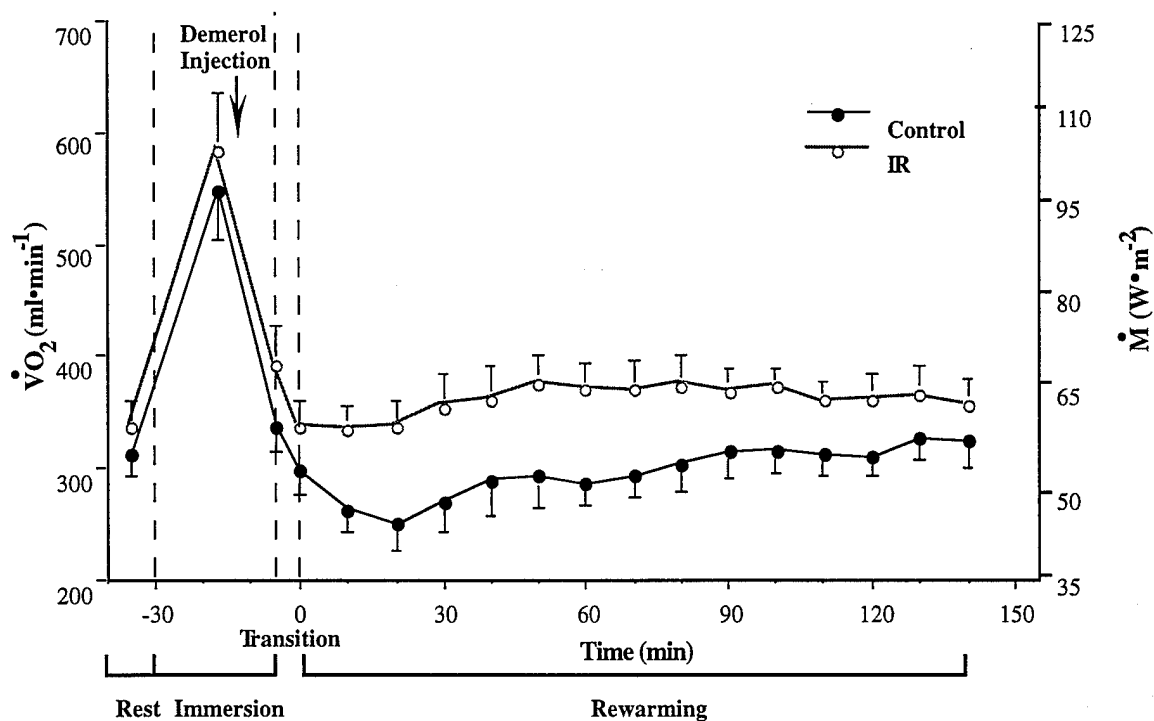


Figure 10. Oxygen consumption ($\dot{V}O_2$) and metabolic heat production (\dot{M}) during the different phases of the Control and Inhalation Rewarming (IR) trials (see Methods for detailed procedures). $n=8$, mean \pm SE.

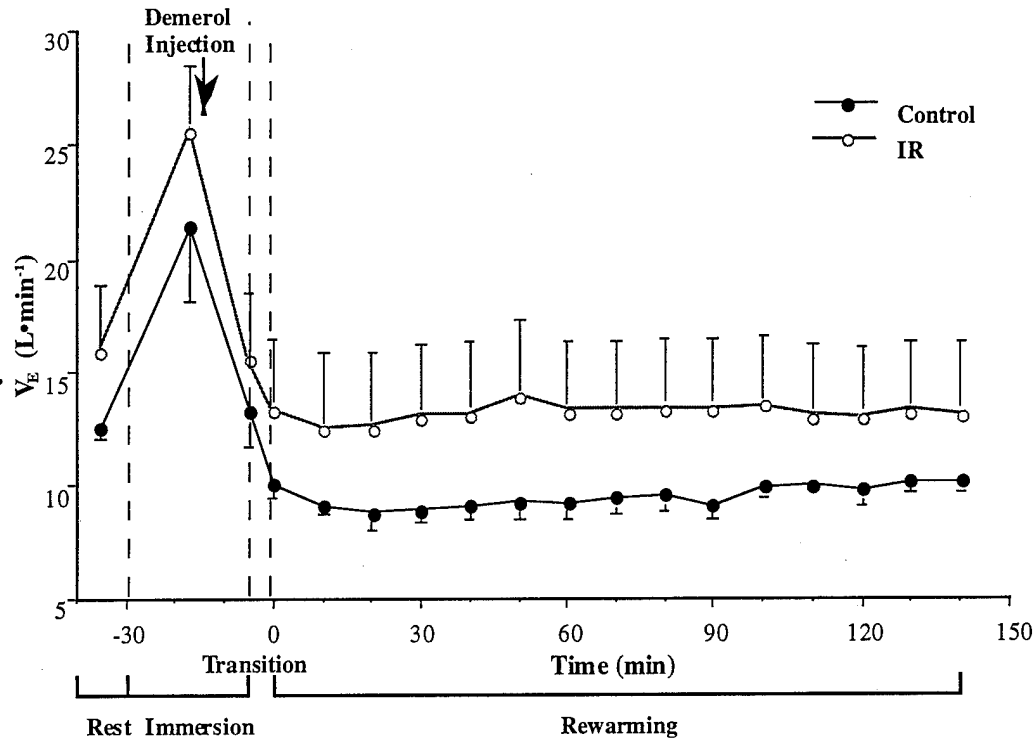


Figure 11. Minute ventilation (\dot{V}_E) observed during the different phases of the Control and Inhalation Rewarming (IR) trials (see Methods for detailed procedures). $n=8$, mean \pm SE.

DISCUSSION

The results from the present study show that when a subject is mildly hypothermic and spontaneous rewarming by shivering is suppressed, inhalation rewarming (humidified air at 43°C) will not significantly reduce the magnitude of the afterdrop, shorten the rewarming period or improve the rate of rewarming when compared to passive rewarming using only the subject's depressed basal metabolism as a heat source. This study only tested the IR treatment on mildly hypothermic subjects because of the limitations of the non-shivering hypothermic model. This artificial condition (mild hypothermia with shivering suppressed) was, however, the only ethically acceptable option that could test the efficiency of the IR treatment on healthy subjects in a laboratory without the interference of shivering as a confounding heat source.

This is the first evaluation, to our knowledge, of an IR treatment on non-shivering hypothermic subjects in the laboratory. Lloyd (1986), however, evaluated inhalation rewarming treatment on deep accidental hypothermic victims with core temperatures ranging between 24.3 and 30°C (mean: 27.4 \pm 0.7°C). No information is provided in the

manuscript regarding the presence of shivering, but since core temperature of the victims was below 30°C, and the victims were unconscious, it may be safe to conclude that shivering was absent during the rewarming phase. Lloyd reported an average rewarming rate of $0.54 \pm 0.03^{\circ}\text{C} \cdot \text{h}^{-1}$ while providing warm humidified air between 50 and 80°C. This rate of rewarming is slightly higher than the rate reported in the present study for the IR trial ($0.23 \pm 0.05^{\circ}\text{C} \cdot \text{h}^{-1}$), possibly because of the higher delivered air temperature in the Lloyd study, but not different from our control trial ($0.41 \pm 0.13^{\circ}\text{C} \cdot \text{h}^{-1}$). On the other hand, most studies using IR treatment on shivering mildly hypothermic subjects reported rates of rewarming for esophageal temperature between 0.8 and $1.4^{\circ}\text{C} \cdot \text{h}^{-1}$ (Romet and Hoskin, 1988; Sterba, 1991; Pozos et al, 1993), which are higher than the rate reported in the present study when shivering was suppressed, but not different from spontaneous rewarming when shivering was not suppressed (Romet and Hoskin, 1988; Sterba, 1991). The difference in the rate of rewarming between these studies and ours is attributed to the contribution of the shivering thermogenesis which can increase the rate of rewarming by 0.5 to $1.1^{\circ}\text{C} \cdot \text{h}^{-1}$, depending on the intensity of shivering. One exception to this general picture is the study of Hayward and Steinman (1975) which reported for one subject a rate of rewarming based on esophageal temperature of $7^{\circ}\text{C} \cdot \text{h}^{-1}$ with an increase in *T_{es}* of 2.1°C during the first 5 min of the treatment and no afterdrop. We suspect that the esophageal probe was misplaced and located in the upper segment of the respiratory tract, and was then directly warmed by the IR system during the treatment. The reported rate of rewarming, therefore, was not an index of the rewarming rate of the subject's core. Regrettably, no other measure of core temperature was included in that study. Finally, other studies reported no differences in the rate of rewarming between IR treatment and passive rewarming (shivering not suppressed) based on tympanic (Collis et al., 1977), ear canal (Marcus, 1978), and rectal (Collis et al., 1977; Marcus, 1978; Harnett et al., 1980; Mekjavic, 1994) temperature measurements.

To explain some of those findings, Lloyd (1973), Shanks and Marsh (1973) and later Sterba (1991) suggested that IR treatment only eliminates respiratory heat loss rather than supplying additional heat. Shanks and Marsh (1973) calculated that IR can improve heat balance by only $23 \text{ kcal} \cdot \text{h}^{-1}$ which is very small considering it is only about 22% of the subject's resting metabolic heat production observed during our IR trials (see Fig. 8), and that humans can increase their heat production by a factor of 5 during vigorous shivering (Iampietro et al., 1960).

One reported advantage of IR treatment over other non-invasive rewarming techniques, particularly peripheral rewarming, is a smaller afterdrop which minimizes the risks of ventricular fibrillation and cardiac arrest, and, therefore, improves the chances of

survival (Harnett, 1983). Several studies reported a smaller afterdrop during the IR treatment when compared to spontaneous rewarming (Lloyd, 1973; Collis et al., 1977; Romet and Hoskin, 1988), but this finding was challenged by others (Harnett et al., 1980; Sterba, 1991; Pozos et al., 1993) including the present study, although we observed a tendency for a lower afterdrop, mainly for *Tear* and *Tre* (see Figs. 2 and 3).

Morrison et al. (1982) and Romet and Hoskin (1988) reported that inhalation rewarming reduces the metabolic heat production in mildly hypothermic subjects (shivering not suppressed), and this reduction was not compensated for by the increased respiratory heat provided by IR. They calculated a reduction of 1.4 kJ (Morrison et al., 1982) and 1.95 kJ (Romet and Hoskin, 1988) of metabolic heat for every 1 kJ of respiratory heat added. In contrast, when shivering is suppressed in hypothermic subjects, the present study shows that IR increased the metabolic heat production by an average of 21% (from 52.4 to 63.4 W • m⁻²; see Fig. 8). This may be at least partly explained by a tendency for the minute ventilation to increase with the IR treatment, possibly because heated and humidified air was not well tolerated by some of the subjects. Two subjects had claustrophobic reactions during the IR trials, while others complained of a burning and suffocating sensation.

With the absence of evident advantages of the IR treatment to efficiently rewarm hypothermic subjects, Sterba (1991) advised to nutritionally support the mildly hypothermic victim in the field to facilitate energy production by shivering, in addition to maximizing insulation to limit convective and conductive heat loss during rescue and pre hospital transport. On the other hand, other authors qualified IR as an effective emergency therapy for accidental hypothermia partly on the basis that it has been used with good results by certain mountain rescue organizations (Collis et al, 1977). Harnett et al. (1983) in their review on hypothermia resuscitation reported several advantages of IR as a first-aid measure, such as 1) the procedure eliminates respiratory heat loss, 2) the technique warms vital organs such as the heart and lungs first, 3) IR thermally stimulates mucociliary activity on the nasopharynx and tracheobronchial tree which may reduce or eliminate the risk of pulmonary edema during hypothermia, and 4) the equipment is simple, portable and inexpensive.

RECOMMENDATIONS

The present study offers no evidence that the IR treatment using the Res-Q-Air inhalation system rewarms faster (higher rate of rewarming) or safer (lower afterdrop) than

spontaneous rewarming when applied on mildly hypothermic non-shivering subjects in simulated field conditions. The advantages of using the IR treatment on mildly hypothermic shivering subjects compared to spontaneous rewarming are also highly controversial based on the literature. Sterba (1991) argued that to show a relevant improvement in the afterdrop or rewarming rate using the IR treatment, one needs to lower the minimum cooling typically used in human experiments (typically 35°C). Further studies should be performed comparing the IR treatment to spontaneous rewarming on more deeply hypothermic shivering subjects (T_{core} around 32-33°C). This condition will allow a greater exchange of heat from the IR system to the tissues because of a higher minute ventilation (more intense shivering due to a lower core temperature) and a larger temperature difference between the air delivered by the IR and the subject's tissues.

Based on the results of the present study, the Res-Q-Air system offered no thermal advantages over spontaneous rewarming to treat hypothermic subjects in simulated field conditions, and we cannot recommend its use by the Canadian Forces at this moment. Further studies are necessary to draw a final conclusion.

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The objective of the present study was to test the efficacy of inhalation rewarming (IR) in simulated field conditions using a newly developed model of non-shivering hypothermia in humans. Eight subjects (2 of them female) were cooled in 8-10°C water for 25 min on two occasions. Ten minutes prior to withdrawal from the water, a total of 1.5 mg•kg⁻¹ of Meperidine, a shivering inhibitor, was injected intravenously into the subjects. The subjects were then removed from the water, settled in a sleeping bag and moved to a cold chamber set at -20°C for a 2.5 hour rewarming period. In the control trial, no external source of heat was available to the subject while in the IR trial, IR was provided (water saturated air at 43°C). In both trials, shivering was successfully suppressed since the metabolic rates during the rewarming phases were not different from baseline values. The duration and magnitude of the post-exposure decrease in esophageal temperature (afterdrop) were not different between the control (78 ± 11 min; 1.4 ± 0.1°C) and the IR trials (76 ± 10 min; 1.2 ± 0.2 °C). The rates of rewarming were not different between the two treatments (0.4 ± 0.1°C • h⁻¹ for control; 0.2 ± 0.1°C • h⁻¹ for IR). After the 2.5 hr rewarming phase, the esophageal temperature did not return to the pre-immersion level for either treatment (35.79 ± 0.17°C for control; 35.85 ± 0.13°C for IR). It was concluded that IR does not decrease the magnitude of the afterdrop and does not enhance rewarming rate in conscious non-shivering hypothermic subjects as compared to no external heat source.

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